The Adverse Effect of Low Levels of Ambient Air Pollutants on Lung Function Growth in Preadolescent Children

Wieslaw Jedrychowski, Elzbieta Flak, and Elzbieta Mróz

Chair of Epidemiology and Preventive Medicine, Collegium Medicum in Jagiellonian University, Kraków, Poland

The main purpose of our study was to assess the effect of low concentrations of ambient air pollution on lung function growth in preadolescent children. We accounted for height velocity over the follow-up period and also for other possible confounders such as baseline anthropometric and physiologic characteristics of children. In addition to outdoor air pollution, we considered the possible effects of social class and exposure to indoor pollutants such as gas stove fumes or environmental tobacco smoke. The cohort prospective study was carried out in 1,001 preadolescent children from two areas of Krakow, Poland, that differed in ambient air pollutants. In the city center (higher pollution area), the mean annual level [± standard deviation (SD)] of suspended particulate matter was 52.6 ± 53.98 μg/m^3 and that of SO_2 was 43.87 ± 32.69 μg/m^3; the corresponding values in the control area were 33.23 ± 35.99 μg/m^3 and 31.77 ± 21.93 μg/m^3. Mean lung function growth rate adjusted to height velocity and lung function level at the study entry was significantly lower in boys and girls living in the more polluted areas. Also, the proportion of children with the slower lung function growth (SLFG) was higher in the children from the more polluted area of the city. The analysis completed in the group of children after the exclusion of asthmatic subjects and those with asthmalike symptoms confirmed that, in boys, odds ratios (ORs) for SLFG [forced vital capacity (FVC)] and air pollution after adjustment to baseline FVC, height, and growth rate was significant [OR = 2.15; 95% confidence interval (CI), 1.25–3.69)]. The analysis also confirmed that for SLFG(FEV_1) the OR was 1.90 (CI, 1.12–3.25). The corresponding OR values in girls were insignificant (OR = 1.50; CI, 0.84–2.68 and OR = 1.39; CI, 0.78–2.44). The association between ambient pollutants and poorer gain of pulmonary volumes in children living in more polluted areas suggests that air pollution in the residence area may be a part of the causal chain of reactions leading to retardation in pulmonary function growth during the preadolescent years. *Key words*: cohort study, epidemiology, lung function growth, outdoor pollution, preadolescent children. *Environ Health Perspect* 107:669–674 (1999). [Online 2 July 1999]
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Annual average concentrations of SO_2 and suspended particulate matter (SPM) exceeding 180–250 μg/m^3 are consistently associated with higher rates of acute and chronic respiratory diseases and are inversely related to lung function (*1–6*). However, as a result of environmental improvement over the past decade, annual mean levels of sulfur dioxide and SPM are currently lower in major cities of Europe, even below 50 μg/m^3; at this level pulmonary effects have not been consistently detected (*7–15*). This may result from the fact that most of the observations have been based on the cross-sectional studies and pulmonary function level measured at one point in time, which reflects the cumulative effects of growth, past and current infections, and physical exercise, together with environmental insults and repair. Mean spirometric values obtained in cross-sectional study for populations with various exposure levels may obscure the real health effects because a minority of a given population may be susceptible to environmental hazard and because these persons may only be identified by repeated spirometric measurements over a period of time.

Longitudinal evaluation involving comparisons of the lung function of the same individual over shorter or longer time and documentation of lung function growth in children may be sensitive indicators of children's current pulmonary health status and possibly to any changes due to a hazardous environment (*16*). The American Thoracic Society (ATS) defined such changes in children as explicit markers of adverse health effects of air pollution indicated by "failure to maintain their predicted lung function growth curve in children" (*17*).

Interpretation of pulmonary function growth in children in terms of environmental hazards is not an easy task because it varies strongly with a child's stage of growth. Cross-sectional analyses have shown that a child's pulmonary function increases linearly with age and height until the adolescent growth spurt, which occurs at approximately 10 years of age in girls and 12 years of age in boys (*18*). Considerable individual variations in the onset and magnitude of the adolescent height growth spurt reported in a number of studies (*19,20*) and the fact that the growth spurt precedes pulmonary function growth by 6–9 months creates additional problems in the interpretation of results (*21*).

The main purpose of our study was to assess the effect of ambient air pollution at low concentrations on the lung function growth in preadolescent children, concentrations that accounted for height velocity over the follow-up period and for other possible confounders such as baseline anthropometric and physiologic characteristics of children. The lung function gain over 2-year follow-up, as measured by forced vital capacity (FVC) and forced expiratory volume in 1 sec (FEV_1), was chosen as the health end point for assessing whether an effect of air pollution is adverse. In addition to outdoor air pollution and the possible effects of social class, exposure to indoor pollutants such as gas stove fumes or environmental tobacco smoke (ETS) were also considered.

Materials and Methods

The study was conducted in Krakow, a city with approximately 700,000 inhabitants, in southern Poland. Daily 24-hr concentrations of suspended particulate matter (black smoke) and SO_2 were obtained from the city network of 17 air monitoring stations run by

Address correspondence to W. Jedrychowski, Chair of Epidemiology and Preventive Medicine, College of Medicine Jagiellonian University, 7 Kopernika Street, Kraków, Poland. Telephone: 48 12 4231-003. Fax: 48 12 4228-795. E-mail: myjedryc@cyf-kr.edu.pl

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the District Sanitary Epidemiological Unit. Annual means were calculated for each pollutant by averaging all available daily values. Subsequently, spatial distribution of air pollutants over the city area was reconstructed using computer modeling.

The spatial distribution of SPM and SO_2 shows that the highest levels of air pollutants were observed in the city center and the lowest were found in the southeastern section of the city. The high-pollution area was defined as the territory within the radius of 0.5 km around each of the four monitoring stations with the highest measurements, whereas the "clean" area was demarcated on a street map within the radius of 0.5 km from each of the two monitoring stations with the lowest air pollution level.

In the city center (higher pollution area), the mean annual level [± standard deviation (SD)] of SPM in 1995 was 52.6 ± 53.98 $\mu g/m^3$ and that of SO_2 was 43.87 ± 32.69 $\mu g/m^3$; the corresponding values in the control area were 33.23 ± 35.99 $\mu g/m^3$ and 31.77 ± 21.93 $\mu g/m^3$. Over the follow-up period the ambient air pollution level was stable in both study areas.

For indoor air quality assessment, three main variables were considered: ETS, system of home heating (gas or coal stove vs. central heating), and the presence of molds on the apartment walls. Exposure to environmental tobacco smoke was expressed as the presence of at least one regular cigarette smoker among parents or guardians of the child. The definition of molds at home was based on mold stains > 1 m^2 on the walls.

For the baseline study, the spatial air pollution distribution in the Krakow area (Figure 1) led to the designation of the children who lived in and attended eight schools located in the highly polluted area as the exposed group and children who lived in and attended six schools in the low-pollution area as the reference group. In the chosen schools a list of students and their residence addresses was prepared; consequently, parents of all 9-year-old children (1,165) that attended the 14 schools were contacted. Of all the parents contacted, 1,129 parents (97% of those eligible) agreed to be interviewed on their household characteristics and the respiratory health of their children. All children lived in the area close to the school; thus, the children living in the high-pollution area attended the schools in the same area and vice versa.

The baseline study was carried out from March through June 1995 and repeated two years later in 1997. The health interviews, performed by trained interviewers, contained questions on respiratory symptoms, as recommended by the epidemiology standardization project (*22*) and the ATS (*23*). Information on household characteristics, ETS, exposure to heavy traffic or industrial low-emission sources located in the residential area, and data on the allergy status of children as diagnosed by a doctor were also requested from the parents. Assessment of body growth (height velocity) was based on the measurements of the standing height of children over a 2-year period (in centimeters). Standing height is assumed to be the most important determinant of pulmonary function growth and height velocity is a reliable index of the onset of puberty during adolescence. For the purpose of the statistical analysis, the children were classified in three categories by their growth rate: fast growth rate, ≥ 14 cm in 2 years; medium growth rate, 10–13 cm; and slow growth rate, < 10 cm).

Spirometric measurements were carried out with a computerized Vitalograph Spirotrac III portable spirometer (Vitalograph, Buckingham, UK). The measurement of air flow from the subject was made using a Fleisch-type pneumotach (Vitalograph). The spirometric testing was done only in the standing position. The best flow of three blows by each child was chosen by the spirometer program according to ATS criteria (*23*). The lung function testing included the FVC and FEV_1 indices. Each day, prior to the lung function examination, the spirometer was checked with a 1-L syringe, and 3% variability was acceptable. All spirometric testing was performed by one trained technician in both health surveys.

The main end point variables of the effects under study was the lung function growth rate as measured by gain in FVC and FEV_1 and the occurrence of slower lung function growth over the 2-year period. Environmental insults during infancy and childhood may pose unusual threats to the respiratory tract because young children are structurally and functionally different from the older children or adults. Many of these differences may increase the vulnerability of young children to air pollutants and hinder postnatal development of the respiratory tract, which may be manifested by slower lung growth. For the purpose of the study, slow lung function growth (SLFG) was defined as whether the gain over 2 years was within the lowest quintile of the distribution of a given test in the particular gender group.

As the first step in the statistical analysis of the data, we carried out the univariate descriptive statistics of pulmonary function indices by outdoor air pollution categories, and the SLFG for each of the spirometric tests was established. Subsequently, multivariate linear regression analyses were used for choosing body variables as significant predictors of the pulmonary growth. Variables that were associated with pulmonary function in this analysis included baseline height and the baseline of a given pulmonary test, growth rate (difference in height between 1997 and 1995), and gender. Because of the skew distribution of lung function indices and growth rate, the variables were transformed into logarithms before being entered

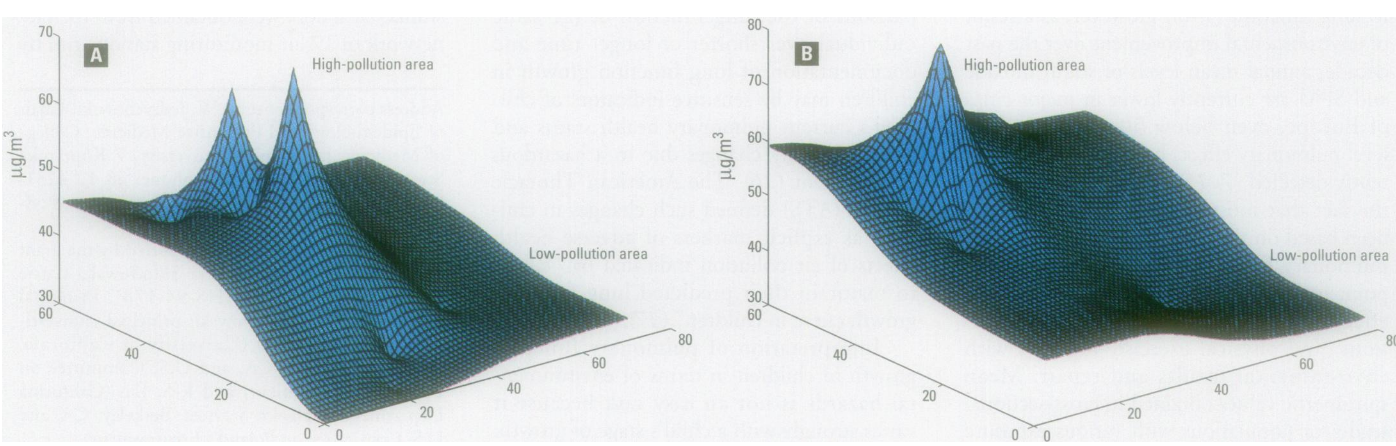

Figure 1. Spatial distribution of (*A*) suspended particulate matter and (*B*) sulfur dioxide in Krakow (1991–1995).

in the multivariate linear models. In the second stage of the analysis, we used multivariate logistic regression (MLR) analysis to calculate the odds ratio (OR) for the SLFG and air pollution adjusted to predictors of lung function growth and other potential confounders (*24*). The following variables were included in MLR models to assess their confounding effect: gender, parental education, ETS, home heating system, and molds on the interior walls. Initially, the confounding variables were considered separately, then all were included in one MLR model to appraise their joint confounding effects. Descriptive and multivariate analysis were done with biomedical computer programs (*25*) separately for boys and for girls.

Results

The population sample studied prospectively over 2 years amounted to 1,001 children (518 boys and 483 girls) of 1,129 children examined at the baseline survey (88.7%). One hundred twenty-eight children were not included in the analysis because of nonparticipation in the second part of the study or because the data from the follow-up were incomplete. Approximately half of the study sample lived in the higher pollution level city center, where coal stoves are more commonly used for home heating. The distribution of exposure to ETS and the social structure of families as defined by parental education were approximately the same in both areas of the city (Table 1).

At the entry to the follow-up, children were 9 years of age (mean ± SD: 8.9 ± 0.4 years of age). Boys were taller than girls (134.4 vs. 133.1 cm) and displayed better lung function at the baseline survey. On average, over the follow-up period boys showed significantly slower growth rate (height velocity) than girls (10.9 vs. 12.0 cm); however, crude values of lung function gain in terms of FVC and FEV_1 did not differ significantly across the gender groups (Table 2).

Table 3 shows that boys who lived in the higher pollution area showed better baseline FVC and FEV_1, but growth rate in boys in this area was significantly lower than in those from the control area (10.2 vs. 11.5 cm); the same has been observed among girls (11.0 vs. 12.9 cm). The fast growth rate (≥ 14 cm in 2 years) was found in 17% of boys and 30.8% of girls, whereas the slower growth rate (< 10 cm in 2 years) was found in 33.2% of boys and 22.6% of girls. The proportion of boys with the slow growth rates was approximately twice as high in the city center as in the low-pollution control area. Among girls, the proportion of children with the slow height velocity was approximately three times higher in the polluted area than in the control area.

Table 1. Characteristics of the study sample by study areas.

Variables	Low pollution	High pollution	Difference χ^2	Difference *p*-Value
Gender of children				
Boys	281 (52.6%)	237 (50.7%)	–	–
Girls	253 (47.4%)	230 (49.1%)	0.350	0.554
Central household heating	524 (98.1%)	265 (56.7%)	255.568	0.000
Molds in household	23 (4.5%)	77 (16.5%)	41.109	0.000
Environmental tobacco smoke	348 (65.2%)	298 (63.9%)	0.200	0.654
Parental education				
Lower	144 (27.0%)	117 (25.1%)	–	–
Higher	390 (73.0%)	350 (74.9%)	0.556	0.456

Table 2. Baseline anthropometry and changes in growth rate and lung function over the study period (1995–1997).

Variables	Boys (mean ± SD)	Girls (mean ± SD)	F	*p*-Value
1995				
Age (years)	8.92 ± 0.403	8.93 ± 0.406	0.29	0.591
Height (cm)	134.35 ± 5.859	133.12 ± 5.847	10.88	0.001
FVC (L)	2.051 ± 0.377	1.866 ± 0.342	65.75	0.000
FEV_1 (L)	1.857 ± 0.333	1.724 ± 0.308	42.87	0.000
1995–1997				
ΔGrowth (cm)	10.91 ± 2.996	12.02 ± 3.285	31.22	0.000
ΔFVC (L)	0.508 ± 0.314	0.487 ± 0.296	1.11	0.291
ΔFEV_1 (L)	0.404 ± 0.274	0.415 ± 0.260	0.43	0.512

Abbreviations: FEV_1, forced expiratory volume in 1 sec; ΔFEV_1, FEV_1 gain over the follow-up; FVC, forced vital capacity; ΔFVC, FVC gain over the follow-up; ΔGrowth, height velocity over the follow-up; SD, standard deviation.

Table 3. Changes in growth rate and lung function over the follow-up in air pollution areas.

Variables	Low-pollution area (mean ± SD)	High-pollution area (mean ± SD)	F	*p*-Value
Boys				
1995				
Age (years)	8.93 ± 0.399	8.91 ± 0.410	0.43	0.510
Height (cm)	133.96 ± 5.760	134.80 ± 5.945	2.61	0.106
FVC (L)	2.011 ± 0.347	2.098 ± 0.405	6.94	0.008
FEV_1 (L)	1.829 ± 0.307	1.892 ± 0.359	4.63	0.032
1995–1997				
ΔGrowth a (cm)	11.47 ± 3.046	10.24 ± 2.797	22.84	0.000
ΔFVC (L)	0.517 ± 0.263	0.497 ± 0.365	0.52	0.471
ΔFEV_1 (L)	0.412 ± 0.246	0.394 ± 0.305	0.60	0.471
Girls				
1995				
Age (years)	8.97 ± 0.407	8.89 ± 0.401	4.71	0.031
Height (cm)	132.91 ± 5.938	133.36 ± 5.750	0.72	0.397
FVC (L)	1.850 ± 0.311	1.884 ± 0.374	1.14	0.286
FEV_1 (L)	1.719 ± 0.281	1.731 ± 0.336	0.18	0.673
1995–1997				
ΔGrowth (cm)	12.91 ± 3.363	11.04 ± 2.900	42.62	0.000
ΔFVC (L)	0.517 ± 0.282	0.455 ± 0.309	5.20	0.023
ΔFEV_1 (L)	0.434 ± 0.245	0.394 ± 0.275	2.84	0.093

Abbreviations: FEV_1, forced expiratory volume in 1 sec; ΔFEV_1, FEV_1 gain over the follow-up; FVC, forced vital capacity; ΔFVC, FVC gain over the follow-up; ΔGrowth, height velocity over the follow-up; SD, standard deviation.

Both boys and girls from the higher pollution area showed lower unadjusted mean values of lung function growth over the study period in terms of FVC and FEV_1 gains than those children who lived in the less-polluted study area (Table 3).

Tables 4 and 5 summarize the results of the multivariate linear regression analysis of the effect of air pollution on lung function growth (FVC and FEV_1) separately in boys and girls. The effect was adjusted for growth rate, baseline height, lung function, and potential cofounders such as ETS, home heating, the presence of molds in the household, and parental education. The results of the analysis demonstrated that there was a significant statistically negative association between air pollution level and lung function growth (FVC and FEV_1) over the follow-up, and this has been confirmed in both gender groups. Lung function growth rate correlated positively with baseline height and growth rate, but negatively with the level of lung function at the follow-up entry. No significant effects of ETS, home heating, the presence of molds in the house, or parental

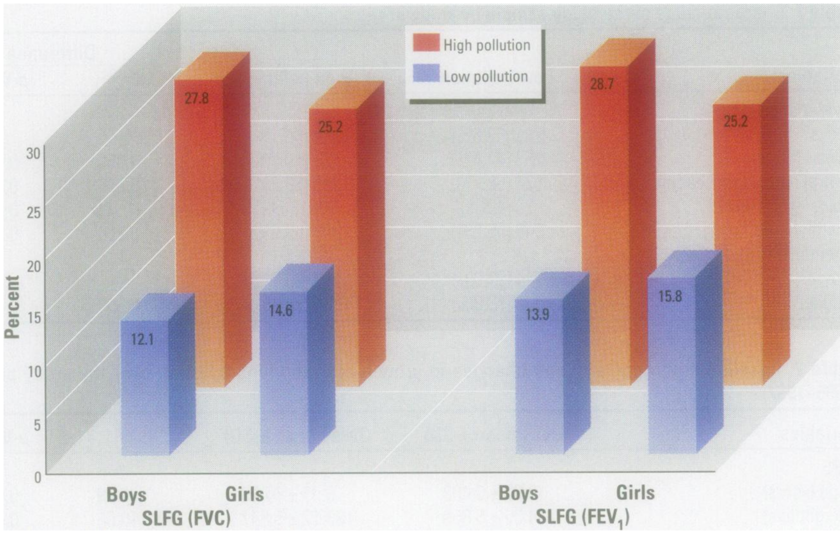

Figure 2. Percentage of children with SLFG over the study period, by air pollution level: the Krakow prospective respiratory study in children. Abbreviations: FVC, forced vital capacity; FEV_1, forced expiratory volume in 1 sec; SLFG, slow lung function growth. Boys: SLFG(FVC) ≤ 0.24 L, SLFG(FEV_1) ≤ 0.18 L. Girls: SLFG(FVC) ≤ 0.23 L, SLFG(FEV_1) ≤ 0.19 L.

Table 4. Regression of log (ΔFVC) related to predictor variables, air pollution, and potential confounders.

Predictors	B-coefficient	SE	*t*	*p*-Value
Boys[a]				
Log height (cm) in 1995	20.864	3.046	6.85	0.00
Log FVC (dL) in 1995	-9.045	0.733	-12.33	0.00
Log growth rate (cm)	0.948	0.412	2.30	0.02
Air pollution[b]	-0.354	0.122	-2.90	0.00
Environmental tobacco smoke[c]	-0.202	0.107	-1.89	0.06
Molds[d]	0.321	0.184	1.74	0.08
Home heating[e]	-0.162	0.146	-1.11	0.27
Parental education[f]	-0.127	0.121	-1.05	0.29
Intercept	-32.881			
Girls[g]				
Log height (cm) in 1995	14.864	2.827	5.26	0.00
Log FVC (dL) in 1995	-7.892	0.669	-11.79	0.00
Log growth rate (cm)	1.123	0.385	2.91	0.00
Air pollution[b]	-0.250	0.108	-2.31	0.02
Environmental tobacco smoke[c]	0.127	0.097	1.31	0.19
Molds[d]	-0.135	0.148	-0.92	0.36
Home heating[e]	0.043	0.129	0.33	0.74
Parental education[f]	0.065	0.106	0.61	0.54
Intercept	-22.440			

Abbreviations: FVC, forced vital capacity; ΔFVC, FVC gain over follow-up; SE, standard error.
[a] $R = 0.5221$, $F_{8,509} = 23.843$, $p < 0.000$. [b] 0, less-polluted area; 1, more-polluted area. [c] 0, nonsmoking parents; 1, at least one smoking parent. [d] 0, no molds; 1, molds present. [e] 0, central heating; 1, coal or gas stoves. [f] 0, lower education of at least one parent; 1, higher education of at least one parent. [g] $R = 0.5093$, $F_{8,474} = 20.755$, $p < 0.000$.

education have been demonstrated on the lung function growth in children.

In the consecutive part of the analysis, SLFG (lowest quintile of distribution of a given spirometric test) was used to display the effect of air pollution. The analysis of the crude data showed that the proportion of children with SLFG was in a clear excess among residents of the more polluted area (Figure 2). Because lung growth rate depends on gender, baseline body size and lung volume level, and height velocity, the multivariate logistic regression model was used in the subsequent analysis to separate the effects of air pollution on lung function from those related to physiologic characteristics of the children. In all logistic models the indoor factors (molds, ETS) and parental education have also been introduced (Tables 6 and 7). The data show that the OR for the occurrence of the SLFG(FVC) and air pollution in boys after adjustment to confounders (height and lung function at baseline survey and body size growth rate) was significant—OR = 2.10 [95% confidence interval (CI), 1.27–3.46]—and that for SLFG(FEV_1) was 2.10 (CI, 1.27–3.48). In girls the corresponding OR values were insignificant: OR = 1.54 (CI, 0.89– 2.64) and OR = 1.51 (CI, 0.90–2.53). In Tables 6 and 7 we did not include the predictors (ETS, molds, parental education) that had trivial impact on the fitting of the statistical model in question.

The analysis repeated in the subsample of 917 children where asthmatic subjects and those with asthmalike symptoms were excluded (asthma diagnosed by physician and/or wheezing independently from respiratory infections and/or dyspnea attacks with wheezing) provided the same results. In boys, ORs for SLFG(FVC) and air pollution after adjustment to baseline FVC, height, and growth rate were 2.15 (CI, 1.25–3.69); ORs for SLFG(FEV_1) were 1.90 (CI, 1.12–3.25). The corresponding OR values in girls were 1.50 (CI, 0.84–2.68) and 1.39 (CI, 0.78–2.44). In all the models the same set of the covariate variables was considered.

Discussion

The results of this study confirmed that even a relatively low air pollution level in the residence area affected lung growth in preadolescent children. We found that the adjusted mean lung function growth rate over the follow-up period was significantly lower among boys and girls living in the more polluted area of the city. The proportion of children with the slower lung growth (SLFG)—irrespective of spirometric test considered—was significantly higher in the more polluted areas only among boys. In girls, there was consistency in the direction of the effect; however, it did not reach the significant level.

The analysis performed in the subsample of children after exclusion of asthmatic subjects (asthma diagnosed by physician and/or wheezing independently from respiratory infections and/or dyspnea attacks with wheezing) confirmed that in boys the adjusted OR for SLFG(FVC) and air pollution was 2.15 (CI, 1.25–3.69), and the adjusted OR for SLFG(FEV_1) was 1.90 (CI, 1.12–3.25); the corresponding OR values in girls were 1.50 (CI, 0.84–2.68) and 1.39 (CI, 0.78–2.44).

The consistent and stable association between ambient pollutants and poorer gain of pulmonary volumes in more exposed children suggests that air pollution in the residence area may be a part of the causal chain of reactions leading to retardation in pulmonary function growth, at least during the preadolescent years. It is difficult to judge whether this is a loss in lung function that would also persist in adolescence or later in adult life. One hypothesis suggests a relationship between childhood respiratory illness and the subsequent development of chronic airflow obstruction in adult life and it is postulated that a decreased level of pulmonary growth in preadolescent children may influence their susceptibility to respiratory infections or environmental insults in adulthood (*26*). If this is so, the identification of risk factors for slower pulmonary function growth and identification of potentially susceptible children should be a key issue in strategies for the early prevention of chronic air flow obstruction.

The level of pollutants within the study areas was rather low, and even in the higher pollution city center the SPM annual mean was 52.6 $\mu g/m^3$ and that of SO_2 was 43.9 $\mu g/m^3$. Therefore, the differences across the areas under comparison were only 19.4 $\mu g/m^3$ for SPM and 12.1 $\mu g/m^3$ for SO_2. These levels of SPM are lower than the hygienic standards proposed by the World Health Organization (WHO) guidelines published in 1987 (*27*); for SO_2 they are also below those recommended in 1997 by WHO (*28*). Because respiratory health of preadolescent children has appeared to be affected even by the low level of SPM in the residence area, our findings support the opinion of the WHO experts that for SPM the hygienic standards should be as low as possible. It is unclear, however, whether the same should be applied for SO_2. Unfortunately, our study dealt with low levels of both SO_2 and SPM in both the city center and within the control area; therefore, we could not differentiate between the effects of SPM and SO_2 on lung function growth.

There is a scarcity of lung function prospective data, particularly in children, related to low air pollution levels. There are data throughout the literature showing significant decrements in lung function that appear to be related to ambient annual total suspended particulate matter > 180 $\mu g/m^3$ (*29*). However, in most of the studies the cross-sectional approach has been used and in many instances the control of confounding has been poor. In addition, differences in results obtained among investigators may arise not only from various research designs but also from the fact that particulate pollution varies in composition and size in different regions and therefore health effects may be related to inhalable and toxic compounds that are concomitant and even not consistently present at all study sites (*30*). In our study, the higher levels of other air pollutants in the city center may have confounded the effect of communal air pollutants; this problem needs more evaluation.

Moreover, our study showed that the growth rates of boys and girls suffered significantly from air pollutants. It is difficult to conclude to what extent the observed effect of air pollution on height velocity in preadolescent children may directly be related to SO_2 or SPM because in the city center higher concentrations of heavy metals and benzo-*a*-pyrene have also been observed. Observations carried out by Perera et al. (*31*) suggest that polycylic aromatic hydrocarbons present in higher concentrations may be responsible for compromising the infant growth in those exposed. However, the slower height velocity in children in the more polluted area of the city strengthens the suggestion that air pollutants may be implicated in compromising both height and lung growth of those exposed.

In our study gas, coal stoves used for household heating, and ETS were not associated with SLFG. The effect of gas stoves on the respiratory function of children has previously been observed (*32*). A substantial body of evidence collected from cross-sectional studies showed that ETS may be associated with the lower lung function level in children (*33–35*); however, some data refute this statement (*36–38*). Our results cannot be directly compared with papers based on cross-sectional designs because we dealt with the dynamics of lung function over a period of time and not with the level of lung function at one point in time. If one assumes that ETS or other indoor pollutants are important determinants of lung function it may be so in the early period of infancy rather than in preadolescence. In the older children, the effect of these factors may be negligible or be overshadowed by the impact of other environmental air pollutants.

Table 5. Regression of log (ΔFEV_1) related to predictor variables, air pollution, and potential confounders.

Predictors	B-coefficient	SE	*t*	*p*-Value
Boys[a]				
Log height (cm) in 1995	18.203	3.288	5.54	0.00
Log FEV_1 (dL) in 1995	-9.481	0.811	-11.69	0.00
Log growth rate (cm)	0.932	0.444	2.10	0.04
Air pollution[b]	-0.307	0.132	-2.33	0.02
Environmental tobacco smoke[c]	-0.137	0.115	-1.18	0.24
Molds[d]	0.274	0.199	1.38	0.17
Home heating[e]	-0.229	0.158	-1.45	0.15
Parental education[f]	-0.041	0.130	-0.31	0.75
Intercept	-27.314			
Girls[g]				
Log height (cm) in 1995	17.728	3.078	5.76	0.00
Log FEV_1 (dL) in 1995	-9.247	0.744	-12.43	0.00
Log growth rate (cm)	1.181	0.424	2.79	0.01
Air pollution[b]	-0.284	0.119	-2.39	0.02
Environmental tobacco smoke[c]	0.182	0.107	1.70	0.09
Molds[d]	-0.139	0.162	-0.86	0.39
Home heating[e]	0.005	0.142	0.03	0.97
Parental education[f]	0.022	0.117	0.19	0.85
Intercept	-27.313			

Abbreviations: FEV_1, forced expiratory volume in 1 sec; ΔFEV_1, FEV_1 gain over the follow-up; SE, standard error.
[a]$R = 0.4937$, $F_{8,509} = 20.508$, $p < 0.000$. [b]0, less-polluted area; 1, more-polluted area; [c]0, nonsmoking parents; 1, at least one smoking parent. [d]0, no molds; 1, molds present. [e]0, central heating; 1, coal or gas stoves. [f]0, lower education of at least one parent; 1, higher education of at least one of parent. [g]$R = 0.5236$, $F_{8,474} = 22.379$, $p < 0.000$.

Table 6. Prevalence OR for SLFG(FVC) related to air pollution category, accounting for potential confounders in the multiple logistic model.

Predictors	OR	CI
Boys[a]		
Height (in cm), 1995	0.86	(0.81–0.91)
FVC (in 100 mL), 1995	1.41	(1.29–1.55)
Growth rate		
10–13 cm	1.12	(0.50–2.47)
< 10 cm	2.61	(1.16–5.89)
Air pollution	2.10	(1.27–3.46)
Girls[b]		
Height (in cm), 1995	0.83	(0.78–0.89)
FVC (in 100 mL), 1995	1.62	(1.44–1.81)
Growth rate		
10–13 cm	3.03	(1.46–6.27)
< 10 cm	5.44	(2.36–12.5)
Air pollution	1.54	(0.89–2.64)

Abbreviations: CI, 95% confidence interval; FVC, forced vital capacity; OR, odds ratio; SLFG, slower lung function growth.
[a]SLFG(FVC) ≤ 0.24 L. [b]SLFG(FVC) ≤ 0.23 L.

Table 7. Prevalence OR for SLFG(FEV_1) related to air pollution category, accounting for potential confounders in the multiple logistic model.

Predictors	OR	CI
Boys[a]		
Height (in cm), 1995	0.87	(0.82–0.92)
FEV_1 (in 100 mL), 1995	1.53	(1.37–1.71)
Growth rate		
10–13 cm	0.81	(0.40–1.68)
< 10 cm	1.58	(0.75–3.33)
Air pollution	2.10	(1.27–3.48)
Girls[b]		
Height (in cm), 1995	0.87	(0.83–0.92)
FEV_1 (in 100 mL), 1995	1.60	(1.43–1.79)
Growth rate		
10–13 cm	2.01	(1.04–3.87)
< 10 cm	3.64	(1.71–7.75)
Air pollution	1.51	(0.90–2.53)

Abbreviations: CI, 95% confidence interval; FEV_1, forced expiratory volume in 1 sec; OR, odds ratio; SLFG, slower lung function growth.
[a]SLFG(FEV_1) ≤ 0.18 L. [b]SLFG(FEV_1) ≤ 0.19 L.

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